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Eveningness associates with lower physical activity from pre- to late adolescence

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Conflicts of Interest

The authors declare that they have no conflict of interest.

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Abstract

Objective: Adolescence is often associated with decline in physical activity (PA) and a circadian shift towards eveningness, but it is not known whether these transitions are intertwined. We explored longitudinally and in cross-section how chronotype and genetic liability for morningness associate with PA as self-reported and measured by actigraphy in early and late adolescence.

Methods: Our sample comes from a longitudinal Finnish community-cohort born in 1998 with information on actigraph-based PA and objectively measured sleep-wake rhythm based on midpoint of sleep at ages 12 (N=353, girls=187) and 17 (N=171, girls=98). Information on self-reported circadian preference and subjective PA was available at age 17. The summarized genetic effects of multiple single nucleotide polymorphism for morningness was assessed by calculating polygenic score (PGS) based on the results on a recent genome-wide association study (GWAS).

Results: PA declined by 40% ($p<0.0001$) in boys and by 32% in girls ($p<0.0001$) from age 12 to 17. Later midpoint of sleep correlated significantly with lower level of general, light and moderate to vigorous PA only at age 12 (all $p<0.05$) but not at age 17 (all $p\geq0.36$). However, those with circadian preference more towards eveningness at age 17 had more sedentary behavior ($p<0.01$) and a lower level of general ($p=0.01$), light ($p<0.01$) and moderate to vigorous PA ($p<0.05$). They also had poorer subjective assessment of their fitness level ($p<0.01$) and they exercised less (all $p\leq0.05$). The decline in objectively measured PA and increase in sedentary behavior from age 12 to 17 was emphasized among those with circadian preference towards eveningness ($p<0.05$). PGS for morningness was not significantly associated with PA in adolescence (all $p\geq0.13$).

Conclusions: Findings of this study highlighted the influence of circadian preference on physical activity behavior in adolescence. Self-assessed circadian preference towards eveningness associated with lower PA and greater decline of it during adolescence. Furthermore, PA declined significantly especially among boys from early to late adolescence. Interventions encouraging physical activity should target specifically evening-oriented adolescents.

Keywords: Chronotype; Eveningness; Exercise; Physical inactivity; Polygenic score; Sleep midpoint

1. Introduction

Physical activity (PA) has a dose-response effect on better adolescent health [1], whereas physical inactivity is a contributing cause to at least 35 unhealthy conditions [2]. However, only less than 20% of the world´s adolescents meet the recommended levels of PA [3]. Adolescence is a period for many biological and behavioral changes. These include, for instance, shifts in circadian rhythms towards eveningness [4–6], which, coupled with early school starts, make adolescents prone to insufficient sleep and daytime sleepiness [7,8].

A marked decline in PA in relation to childhood is common in adolescence [9–11]. A previous British study showed a yearly ~10% increase in sedentary behavior from age 10 to 14, corresponding to an over 40 minutes decrease in daily PA [9]. During adolescence, there is also a progressive shift towards more evening-oriented circadian rhythm [4,6], influenced by genetic tendencies [12], hormonal changes [13], and by changes in the psychosocial environment [13,14]. However, it is not known whether these two transitions, i.e. decline in PA levels and shift towards eveningness, are intertwined. Some evidence exists showing that morning-oriented adolescents report exercising more than evening-types or those with late bedtimes [15–20]. With regard to objectively measured PA, both later midpoint of sleep and circadian preference towards eveningness associate with less moderate to vigorous PA (MVPA) and increased sedentary behavior in adults [21,22]. It is still unclear on whether sleep midpoint or genetic tendency to morningness/evenigness associate with objective or subjective PA and its changes in adolescence.

1.1 Current Study

Thus far, studies on individual factors affecting trajectories of physical activity across adolescence is scarce. Accordingly, the objective of this study was to examine objectively and subjectively measured PA levels in relation to self-reported circadian preference, midpoint of sleep defined by an actigraph, and genetic tendency towards morningness. For this purpose, we used a polygenic score (PGS) for morningness based on the recent genome-wide association study (GWAS) on the subject [23]. These associations were studied both longitudinally and in cross-section from early (12 years) to late (17 years) adolescence in a sample derived from a Finnish cohort study. Based on prior research, we hypothesized that eveningness would associate with less subjective and objective PA and with a greater decline of PA from early to late adolescence.. In addition, we examined the difference in PA change between boys and girls as previous studies indicate that a greater decline in PA among boys than among girls [10].

2. Material and Methods

2.1 Participants

The analytic sample used here is based on adolescent follow-ups from a Finnish urban community-based cohort [4]. At T1, when participants were 12 years of age on average, genetic samples were extracted and actigraph-based physical activity and sleep were measured. At T2, when participants were 17 years of age on average, participants reported their circadian preference and exercise habits and actigraphy was used to measure daytime physical activity and sleep patterns.

The analytic sample at T1 (N=353, girls=187, boys=166; Mean age=12.3, SD=0.5 years) consisted of those with information on actigraph-based physical activity and sleep measurement for at least 2 days at T1 and a genetic sample extracted at T1. The analytic sample at T2 (N=172, girls=99, boys=73; Mean age=16.9, SD=0.1 years) consisted of those with information on actigraph-based physical activity and sleep measurement at T2 for at least 2 days, self-reported circadian preference at T2 and a genetic sample extracted at T1. Longitudinal sample consisted of 167 individuals with complete data from both T1 and T2 (56.4% girls).The Ethics Committee for Children and Adolescents' Diseases and Psychiatry at the Helsinki University Central Hospital approved the study protocol. All participants and their parents gave their written informed consent.

2.2. Objective measurement of physical activity and sleep-wake rhythm by actigraphy at T1 and T2

The daytime PA and sleep patterns at T1 and T2 were measured with actigraphs (Actiwatch AW7, Cambridge Neurotechnology Ltd., UK) worn on the non-dominant wrist with a 1-minute epoch length.. For PA, at least 10 hours of PA data per day from 9AM onwards was considered a valid day recording [24]. A minimum of at least two valid days/nights recording was required per participant to be included in the analyses (T1 Mean=7.99, SD=1.76 measured days; T2 Mean=6.91, SD=2.23 measured days).

We assessed LPA, MVPA and sedentary behavior as average percentages per day by dividing the cpm for each activity measurement day. General PA at T1 and T2 was calculated as the total counts per minutes (cpm) and averaging this over the measurement period for each participant. Sedentary behavior was calculated as activity below 320 cpm, light PA (LPA) as activity from 321 to 1047 cpm and moderate to vigorous activity (MVPA) as activity from 1048 to above according the appropriate metabolic equivalents based on a validation study on children regarding the same actigraph used here [25].

For sleep measurement (T1 Mean=8.10, SD=1.73 and T2 Mean=8.12, SD=1.97 measured nights), the participants were instructed to report the wake-up time and bedtime by pressing an event marker button in the

actigraph device as well as by keeping a sleep log., that helped to discard any nights with significant confounding (eg., sickness, travel). Average midpoint of sleep was determined by the half of the time passed in sleep between the sleep onset and waking up. To define sleep quality, we used wake after sleep onset (WASO) time, calculated as the amount of minutes recorded as wake after the sleep onset. The actigraph measurement periods -included both weekdays and weekends.

2.3 Change in general PA from T1 to T2

The change in general PA from T1 to T2 was calculated as a difference score (T2-T1), a smaller score indicating a larger decline in PA. In our sample, there were 10.2% of participants (N=17) whose PA level increased from T1 to T2. PA declined from T1 to T2 in 89.8% of the participants (N=150). We split these participants into three groups: 1) increasers in PA (N=17, mean difference between T1 and T2 (MD)=493.88 cpm, SD=1813.87), 2) moderate decliners in PA (N=75, median split among the declining group, MD=-85.62, SD=40.60) and 3) large decliners in PA (N=75, MD=-259.50, SD=90.66).

2.4 Self-reported exercising habits at T2 (age 17)

Self-reported PA was assessed with the following five questions at age 17: 1) Describe your fitness level (from 1=very poor to 5=very good); 2) How often do you exercise in your free-time? (from 1=not at all to 7=almost every day); 3) How long does your average exercise time last? (1=I do not exercise to 5=from two hours to longer); 4) How much light exercise do you do at your free-time? (1=not at all to 6=about seven or more hours a week); 5) How much brisk exercise do you do at your free-time? (1=not at all to 6=about seven or more hours a week). These self-reported items were used as continuous indicators of PA.

2.5 Subjective circadian preference assessment

Participants reported their chronotype at T2. To create consistency with the design in the GWAS [23] utilized for PGS calculation here, we used item 19 from Morningness-Eveningness Questionnaire (MEQ [26]) to define circadian preference. This item correlates significantly with the full MEQ in our sample (r=0.781, p<0.0001). The question requests individuals to estimate their circadian preference as either 1=Definitely a ‘morning’ person, 2=More a ‘morning’ than an ‘evening’ person, 3=More an ‘evening’ than a ‘morning’ person or 4=Definitely an ‘evening’ person. Similarly to GWAS on circadian preference [23], we assessed this item as continuous in other to calculate the best fit polygenic score for morningness by scoring those answering Definitely a ‘morning’

person=2, More a ‘morning’ than an ‘evening’ person=1, More an ‘evening’ than a ‘morning’ person=-1 and Definitely an ‘evening’ person=-2.

2.6 Genotyping and calculation of the polygenic score for morningness

DNA was extracted from blood (22%) and saliva samples (78%) collected at the 2009-2011 follow-up and the genotyping was performed with the Illumina OmniExpress Exome 1.2 bead chip at the Tartu University, Estonia, in September 2014, according to the standard protocols. IMPUTE2 software and the 1000 Genomes Phase I integrated variant set (v3 / April 2012; NCBI build 37 / hg19) as the reference sample was used for imputation. Before imputing the following quality control filters were applied: single nucleotide polymorphism (SNP) clustering probability for each genotype >95%, call rate >95% individuals and markers (99% for markers with minor allele frequency (MAF) <5%), MAF>1 %, Hardy–Weinberg equilibrium (HWE) p>1×10-6.

The polygenic score (PGS) for morningness was calculated based on a recent GWAS [23] across whole genome. PGS for morningness was computed using the beta and p-values from summary statistics of this GWAS for self-reported chronotype, based on data from 128,266 British adults, aged 37 to 73 years, from the UK Biobank study and replicated in 89,283 23andMe participants [23]. Before calculating the PGS, clumping was performed with plink 2.0 (http://www.cog-genomics.org/plink/2.0/) [27] in order to remove the SNPs which are in linkage disequilibrium with each other (r^2 value 0.1 used according to the default value in PRSice software). For the PGS analysis, the statistical analysis software package PRSice v2.2.0 was used [28]. Best fit PGS for genetic tendency towards morningness was gained with a p-value threshold of 0.0001 including 354 SNPs.

2.7 Assessment of pubertal stage and BMI

Pubertal maturation was self-assessed at T1 and T2 with the Pubertal Developmental Scale (PDS) [29] as described previously. The PDS scale is 5-item self-report scale concerning body hair, growth spurts, skin changes, for girls menarche and breast development, for boys facial hair and voice change scored 1=no changes to 4=development complete. Body mass index (BMI; kg/m^2) was measured at the clinical visit at T1 and during the nurse´s home visit at T2.

2.8 Statistical analysis

We used correlation analysis to analyze how self-reported circadian preference, sleep midpoint and PGS towards morningness were related. As previous study on the same sample at T1 showed that physical activity differed

between sexes [30], one-way ANOVA was used to analyze the differences between sex in actigraph-based PA at T1 and T2, midpoint of sleep, subjective circadian preference, genetic tendency towards morningness, BMI and pubertal status. Repeated measures ANOVA was used to analyze the difference in PA from T1 to T2 separately by sex.

We used hierarchical regression analyses to analyze the association between circadian timing and actigraph-based PA at T1 and T2. For the associations at T1, sex, age and pubertal status were included in the model in the first step, sleep midpoint at age 12 in the second step and PGS for morningness in the final step. For the associations at T2, sex, age and pubertal status were included in the model in the first step, self-reported circadian preference in the second step, sleep midpoint at age 17 in the third step, and PGS for morningness in the final step. General linearized models adjusted with sex and age were used to analyze the association between circadian preference, sleep midpoint or PGS for morningness and self-reported exercising habits at age 17. Finally, hierarchical regression analyses were used for the association between circadian timing and change in actigraph-based PA from T1 to T2. Sex and changes in age and pubertal status from T1 to T2 were included in the model in the first step, self-reported circadian preference in the second step, change in sleep midpoint from T1 to T2 in the third step, and PGS for morningness in the final step.

3. Results

3.1 Initial analyses of the study sample

The analytic sample of participants at T1 and/or T2 in this study did not differ significantly from the rest of the participants in the initial cohort regarding gestational age, birthweight, length at birth or maternal alcohol consumption during pregnancy in T-tests (all $P>0.15$).

As reported previously for the same cohort [12], self-reported circadian preference towards morningness at T2 correlated with higher genetic predisposition for morningness ($r=0.16$, $p=0.03$) and with earlier timed midpoint of sleep at T2 ($r=-0.33$, $p<0.0001$). Midpoint of sleep at T1 and T2 did not correlate significantly with the PGS for morningness ($p>0.08$ and $p>0.06$, respectively).

As Table 1 shows, girls were significantly further ahead in pubertal development than boys in our sample at both T1 and T2. There were no significant differences in BMI between boys and girls at T1 or T2. As shown in Table 1 and Figure 1, at T1, boys were more active than girls. Boys had a higher level of general PA (Figure1a) and a higher percent of MVPA, but less LPA (Figure1b) than girls. Girls and boys had an equal share of sedentary time during the day (Figure1b). At T2, boys had more sedentary time and less LPA than girls

(Figure1b). There were no significant differences between boys and girls in general PA and MVPA at age 17. Overall, general PA declined by 32% from T1 to T2 among girls ($p=0.0001$) and by 40% among boys ($p<0.0001$), while sedentary behavior increased from T1 to T2 by 25% among girls ($p<0.0001$) and by 32% among boys ($p<0.0001$). LPA declined by 25% from T1 to T2 among girls ($p<0.0001$) and by 30% among boys ($p<0.0001$). MPA declined by 55% from T1 to T2 among girls ($p<0.0001$) and by 63% among boys ($p<0.0001$). As illustrated in Figure 1a, the decline in general PA from T1 to T2 was significant among boys.

As Table 1 shows, midpoint of sleep at T2 was significantly later among boys than among girls by 30 minutes. There were no significant differences between sexes in midpoint of sleep at T1, self-reported circadian preference at T2, or in the PGS for morningness ($p=0.38$). Sleep duration did not differ significantly by sex at T1, but at T2 boys slept by average 14 minutes less than girls. Sleep quality was somewhat poorer in boys at T1, with 5 minutes more WASO as compared to girls. There were no differences in sleep quality between sexes at T2.

3.2 Actigraphy-based PA by chronotype at the age 12 (T1)

As Table 2 shows, later midpoint of sleep at T1 correlated significantly with lower level of general PA, more sedentary behavior, and less LPA and MVPA. PGS for morningness and PA at T1 did not correlate significantly. Table 3 shows the hierarchical regression models for PA at T1. Age was significantly associated with PA explaining together with sex and pubertal status 6% to 13% of variance in PA, while sex associated with LPA and MVPA. Introducing either midpoint of sleep at T1 in the second step or PGS for morningness in the third step did not contribute significantly to the models.

3.3 Actigraphy-based PA and self-reported exercising habits by chronotype at the age 17 (T2)

As Table 2 shows, those with circadian preference towards eveningness at T2 had less general PA, LPA and MVPA and more sedentary behavior than those with circadian preference towards morningness. Midpoint of sleep or PGS for morningness did not significantly correlate with PA at T2.

As Table 4 for the hierarchical regression models at T2 shows, sex, age and pubertal status were not significantly associated with PA at T2. The second step, introducing self-reported circadian preference, contributed significantly to models on general PA, LPA and sedentary behavior, explaining additional 4 to 5% of variance in PA. Introducing midpoint of sleep T2 in the third step or PGS for morningness in the final step did not significantly add to the explained variance of PA variables. Those with circadian preference towards morningness

reported having better fitness level (β=0.13, 95% CI=0.04 to 0.23, p=0.006), they exercised more at their free-time (β=0.28, 95% CI=0.13 to 0.44, p=0.0004), had longer workouts (β=0.13, 95% CI=0.03 to 0.23, p=0.009) and had more both light (β=0.18, 95% CI=0.03 to 0.33, p=0.02) and vigorous (β=0.33, 95% CI=0.17 to 0.50, p=0.00007) PA at their free-time than those with circadian preference towards eveningness. Midpoint of sleep at (all p>0.09) and PGS for morningness (all p>0.20) were not significantly associated with self-reported PA.

3.4 Change in actigraphy-based PA from age 12 to age 17 by chronotype

In hierarchical regression analyses, sex, time between T1 and T2, and pubertal status explained together 8% of variance in PA change from T1 to T2. Of these, only sex was significantly associated with PA change indicating that boys had a greater decline in PA than girls from T1 to T2 (β=-0.23, p=0.008, for age and pubertal status p-values>0.11). The second step, introducing self-reported circadian preference, explained an additional 4% of variance in PA change. Those with circadian preference towards eveningness had a larger decline in PA (β=0.20, p=0.02) than those with circadian preference towards morningness. Introducing change in midpoint of sleep from T1 to T2 in the third step or PGS for morningness in the final step did not significantly add to the explained variance of change in PA (p>0.69 and p>0.95 respectively). Figure 2 shows the mean circadian preference by general PA change trajectories. It shows that eveningness was more prevalent in both PA decline groups.

4. Discussion

Many studies have found a declining trajectory of physical activity during adolescence [9–11]. However, less is known of factors individually affecting these trajectories. We examined this question by studying objective and subjective chronotype markers that included self-reported circadian preference, actigraph-based sleep midpoint, and a polygenic score for genetic tendency towards morningness, in relation to the level and development of PA from age 12 to 17. For PA measurements, we used both actigraphy-based daytime physical activity levels at both ages, and complemented the assessment with aself-reported fitness level and exercising habits at age 17.

At age 12, later actigraphy-based midpoint of sleep correlated with lower daytime PA, but five years later, at age 17, there was no significant association between PA or midpoint of sleep. However, at age 17, those with circadian preference towards eveningness had significantly more sedentary behavior and a lower level of general PA, LPA and MVPA than those with a circadian preference towards morningness. Furthermore, the decline in objectively measured PA and increase in sedentary behavior from age 12 to age 17 was emphasized among those with self-reported circadian preference more towards eveningness. Objective chronotype markers,

sleep midpoint and PGS for morningness, did not explain any additional variance in the developmental trajectories of PA, and PSG for morningness was not associated with daytime PA during adolescence.

As expected, there was a moderate association between all the chronotype markers used here: those with circadian preference more towards eveningness had later midpoint of sleep and lower genetic tendency for morningness. These chronotype variables do not, however, represent exactly the same phenomenon as circadian preference describes what the individual would feel as their optimal behavioral timing, while midpoint of sleep describes more of the actualized sleep-wake rhythm for a certain time period which can be subject to factors other than the individual´s preference. Genetic tendency, on the other hand, does not necessary translate to the individuals actualized phenotype, as almost 50% of the individual´s chronotype is estimated to be influenced by environmental factors [31]. Furthermore, the participants of the GWAS on which the PGS for morningness is based, were 40 to 69 year old adults in the UK Biobank sample [23]. It is thus possible that calculating PGS based on a different population with its own genetic makeup and from a sample with different age range influences how well suited the PSG is in capturing chronotype in the current study. Regardless, our analyses indicated that PGS for morningness and objectively assessed midpoint of sleep had did not echo the effect of later circadian preference on physical activity behavior. . Supporting the value of using subjective circadian preference as a circadian typology marker, previous studies have shown that eveningness based on full and reduced MEQ score has been shown to associate with shorter and later-timed activity acrophase as compared to morningness [32–34].

While there is lack of similar studies with objective sleep and PA indicators to compare our results, previous study on 2200 adolescents between ages 9 to 16 years is aligned with our results by showing that those reporting late bed-/rise time rhythm had less MVPA than those reporting early bed-/rise times unrelated to sleep duration [35]. Similarly, circadian preference towards eveningness has been associated with more sedentary behavior and less PA than circadian preference for morningness in adults [21]. Also similarly to previous findings in adults [22], later midpoint correlated with lower PA at age 12 in our study. However, it did not correlate with PA at age 17, and later midpoint did not add explained variance of PA in any models.

Regarding general behavioral habits and self-estimated physical condition, our results also showed that those with circadian preference more towards eveningness self-reported poorer fitness level and exercising habits. These results are in line with previous studies on self-reported physical behavior and chronotype suggesting that circadian preference towards eveningness associates with higher physical inactivity in adolescents [16,20], less exercising among both adolescents [15,18] and adults [17] and shorter physical exercise time in young adults [36] than circadian preference towards morningness.

It is possible that those with circadian preference towards eveningness have lower PA because they have more difficulties finding a suitable time in line with their preference to exercise. Studies on adult psychophysiological responses to physical activity in different chronotypes have shown that Evening-types feel more exhausted and their physical performance is poorer when physical activity is timed early in the day rather than in the evening [37]. Previous studies confirm that those with circadian preference towards eveningness have also later actigraph-based activity acrophase than those with circadian preference towards morningness [34,38] highlighting difference in the peak performance time among circadian preference types. It is also possible that Evening-types have less energy to exercise due to higher amount of sleep difficulties than those with circadian preference towards morningness [7,8]. Previous studies have shown that adolescents with late bedtimes not only exercise less but also report more sleep problems, day-time tiredness and higher accident propensity than those with earlier bedtimes [18]. Eveningness predisposes adolescents to sleep problems as school and other social schedules during weekdays are not in line with the sleep-wake and other behavior timing preferences of those with circadian preference more towards eveningness. This has been shown in a previous study indicating that those with circadian tendency more towards eveningness accumulate sleep debt during school/working days creating a social jetlag [39]. Lack of sleep on the other hand has been shown to associate with low levels of PA in adolescents [40,41].

Adolescents in this study met on the average the recommended amount of 60 minutes of moderate to vigorous PA per day [3] at both T1 and T2. In general, adolescents in our sample slept on average at age 12 very close to the lower limit of the recommended 8 to 10 hours per night for teenagers and at age 17 in the border of necessary sleep duration per night [42]. However, both at age 12 and 17 sleep quality in our sample was not optimal as WASO exceeded 20 minutes on average [43]. Boys had poorer sleep quality at age 12 and they had shorter sleep than girls at age 17. Shorter sleep duration than recommended combined with poor sleep quality among boys might at least partly explain why the decline in objectively measured physical activity in our study from age 12 to 17 was larger among boys (-40%) than girls (-32%). Previous studies on adolescent PA [10] are in line with our finding of greater decline in PA among boys than among girls. In turn, sedentary activity increased from age 12 to age 17 by 32% in boys and 25% in girls. The mean yearly change in boys (-8% per year) was close to that reported before in a meta-analysis of 26 studies (7% [10]). For girls, the mean yearly change was -6.4%, which is almost the same as reported elsewhere (-6.3% [10]). Girls were ahead of boys in pubertal status at each time point, which could explain why PA levels were lower for girls at T1 as compared to boys and be another explanation for why the decline in PA between T1 and T2 was more drastic in boys than in girls. Pubertal

development in girls has been associated with lower level of physical activity and indicated to be mostly due to changes in psychological well-being, such as increased depression and negative self-awareness [44]. Pubertal status has also been suggested to influence more than chronological age on both physical activity level as well as the circadian shift towards eveningness during adolescence in girls [45]. Similar negative effects of pubertal maturation on PA in boys have also been previously reported, where PA has declined more drastically in boys with early pubertal maturation than in later-maturing boys [46].

4.1 Limitations and strengths

As a limitation, we did not have data on the subjective circadian preference at the age of 12, and self-reported PA was available only at the age 17. Actigraph-based measurements were not all performed at the same time of the year at T1 or at T2. We also lacked information on actigraph-based rest activity circadian rhythm, which would have been an interesting addition to the study. Strengths of our study include the large longitudinal sample of actigraph-based PA at the ages 12 and 17, which enable examining the change in PA with age in relation to chronotype. Other strengths of our study include differentiation between chronotype indicators of genetic tendency, actualized behavior and behavioral preference.

5. Conclusions

PA levels during childhood and adolescence determine to large extent adult physical activity levels, and therefore health prospective [47]. The results of this study emphasize the role of subjective circadian preference as the primary indicator relevant for understanding variation and developmental trajectories of PA in adolescence. Eveningness associated with not only poorer subjective fitness level and exercising habits but also, as a novel finding, with lower levels of objectively measured PA during adolescence. Additionally, our study shows that circadian preference towards eveningness associated with negative development in PA level from early to late adolescence. Physical inactivity has many negative health consequences, not only in adulthood [2] but also during adolescence [1,48]. Not surprisingly, population-based studies indicate that the same health risks related to physical inactiveness [2] are also more prevalent among adults with circadian preference towards eveningness [17,49,50]. These findings may offer new perspectives to understand developmental aspects of this phenomenon. Furthermore, these findings should encourage interventions in scheduling physical activities to better motivate physical activity among evening-oriented adolescents. The timing of the physical activity might play a key role in

motivating an evening-oriented adolescent to exercise more, but also ensuring long enough sleep at night is essential in preventing daytime tiredness.

References

[1] Janssen I, LeBlanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. Int J Behav Nutr Phys Act 2010;7:40. https://doi.org/10.1186/1479-5868-7-40.

[2] Booth FW, Roberts CK, Thyfault JP, Ruegsegger GN, Toedebusch RG. Role of Inactivity in Chronic Diseases: Evolutionary Insight and Pathophysiological Mechanisms. Physiol Rev 2017;97:1351–402. https://doi.org/10.1152/physrev.00019.2016.

[3] WHO. Global status report on noncommunicable diseases 2010: description of the global burden of NCDs, their risk factors and determinants. WHO 2011.

[4] Kuula L, Pesonen A-K, Merikanto I, Gradisar M, Lahti J, Heinonen K, et al. Development of Late Circadian Preference: Sleep Timing From Childhood to Late Adolescence. J Pediatr 2018;194:182-189.e1. https://doi.org/10.1016/j.jpeds.2017.10.068.

[5] Merikanto I, Pesonen A-K, Kuula L, Lahti J, Heinonen K, Kajantie E, et al. Eveningness as a risk for behavioral problems in late adolescence. Chronobiol Int 2017:1–10.

[6] Roenneberg T, Kuehnle T, Pramstaller PP, Ricken J, Havel M, Guth A, et al. A marker for the end of adolescence. Curr Biol 2004;14:R1038–9.

[7] Gradisar M, Gardner G, Dohnt H. Recent worldwide sleep patterns and problems during adolescence: a review and meta-analysis of age, region, and sleep. Sleep Med 2011;12:110–8.

[8] Touitou Y. Adolescent sleep misalignment: a chronic jet lag and a matter of public health. J Physiol-Paris 2013;107:323–6.

[9] Corder K, Sharp SJ, Atkin AJ, Griffin SJ, Jones AP, Ekelund U, et al. Change in objectively measured physical activity during the transition to adolescence. Br J Sports Med 2015;49:730–6. https://doi.org/10.1136/bjsports-2013-093190.

[10] Dumith SC, Gigante DP, Domingues MR, Kohl HW. Physical activity change during adolescence: a systematic review and a pooled analysis. Int J Epidemiol 2011;40:685–98. https://doi.org/10.1093/ije/dyq272.

[11] Farooq MA, Parkinson KN, Adamson AJ, Pearce MS, Reilly JK, Hughes AR, et al. Timing of the decline in physical activity in childhood and adolescence: Gateshead Millennium Cohort Study. Br J Sports Med 2018;52:1002–6. https://doi.org/10.1136/bjsports-2016-096933.

[12] Merikanto I, Lahti J, Kuula L, Heinonen K, Räikkönen K, Andersson S, et al. Circadian preference and sleep timing from childhood to adolescence in relation to genetic variants from a genome-wide association study. Sleep Med 2018;50:36–41. https://doi.org/10.1016/j.sleep.2018.04.015.

[13] Crowley SJ, Acebo C, Carskadon MA. Sleep, circadian rhythms, and delayed phase in adolescence. Sleep Med 2007;8:602–12. https://doi.org/10.1016/j.sleep.2006.12.002.

[14] Harbard E, Allen NB, Trinder J, Bei B. What's Keeping Teenagers Up? Prebedtime Behaviors and Actigraphy-Assessed Sleep Over School and Vacation. J Adolesc Health 2016;58:426–32. https://doi.org/10.1016/j.jadohealth.2015.12.011.

[15] Kauderer S, Randler C. Differences in time use among chronotypes in adolescents. Biol Rhythm Res 2013;44:601–8. https://doi.org/10.1080/09291016.2012.721687.

[16] Malone SK, Zemel B, Compher C, Souders M, Chittams J, Thompson AL, et al. Characteristics Associated With Sleep Duration, Chronotype, and Social Jet Lag in Adolescents. J Sch Nurs 2016;32:120–31. https://doi.org/10.1177/1059840515603454.

[17] Merikanto I, Englund A, Kronholm E, Laatikainen T, Peltonen M, Vartiainen E, et al. Evening chronotypes have the increased odds for bronchial asthma and nocturnal asthma. Chronobiol Int 2014;31:95–101. https://doi.org/10.3109/07420528.2013.826672.

[18] Merikanto I, Lahti T, Puusniekka R, Partonen T. Late bedtimes weaken school performance and predispose adolescents to health hazards. Sleep Med 2013;14:1105–11. https://doi.org/10.1016/j.sleep.2013.06.009.

[19] Schaal S, Peter M, Randler C. Morningness-eveningness and physical activity in adolescents. Int J Sport Exerc Psychol 2010;8:147–59.

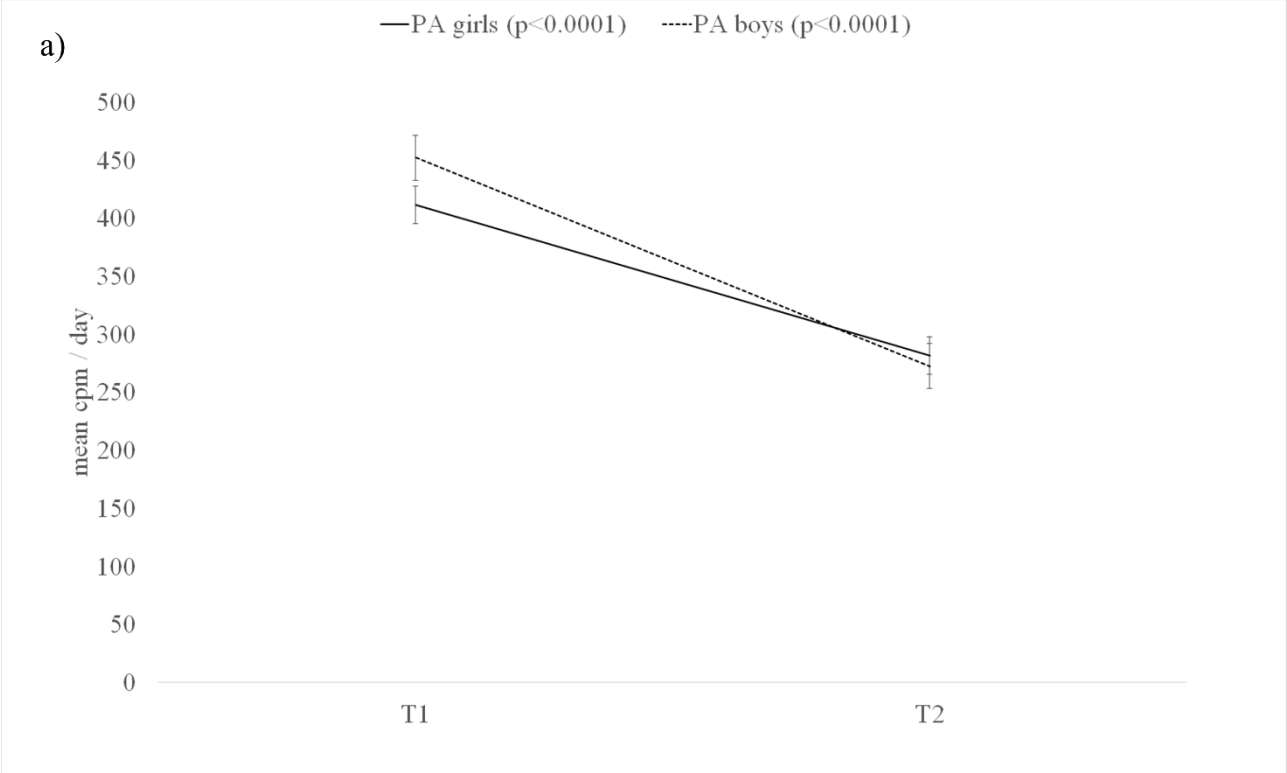
[20] Urbán R, Magyaródi T, Rigó A. Morningness-Eveningness, Chronotypes and Health-Impairing Behaviors in Adolescents. Chronobiol Int 2011;28:238–47. https://doi.org/10.3109/07420528.2010.549599.

[21] Hisler GC, Phillips AL, Krizan Z. Individual Differences in Diurnal Preference and Time-of-Exercise Interact to Predict Exercise Frequency. Ann Behav Med 2017;51:391–401. https://doi.org/10.1007/s12160-016-9862-0.
[22] Shechter A, St-Onge M-P. Delayed sleep timing is associated with low levels of free-living physical activity in normal sleeping adults. Sleep Med 2014;15:1586–9. https://doi.org/10.1016/j.sleep.2014.07.010.
[23] Jones SE, Tyrrell J, Wood AR, Beaumont RN, Ruth KS, Tuke MA, et al. Genome-Wide Association Analyses in 128,266 Individuals Identifies New Morningness and Sleep Duration Loci. PLOS Genet 2016;12:e1006125. https://doi.org/10.1371/journal.pgen.1006125.
[24] Matthews CE, Hagströmer M, Pober DM, Bowles HR. Best practices for using physical activity monitors in population-based research. Med Sci Sports Exerc 2012;44:S68.
[25] Ekblom O, Nyberg G, Bak EE, Ekelund U, Marcus C. Validity and Comparability of a Wrist-Worn Accelerometer in Children. J Phys Act Health 2012;9:389–93. https://doi.org/10.1123/jpah.9.3.389.
[26] Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol 1975;4:97–110.
[27] Chang CC, Chow CC, Tellier LC, Vattikuti S, Purcell SM, Lee JJ. Second-generation PLINK: rising to the challenge of larger and richer datasets. GigaScience 2015;4. https://doi.org/10.1186/s13742-015-0047-8.
[28] Euesden J, Lewis CM, O'Reilly PF. PRSice: Polygenic Risk Score software. Bioinforma Oxf Engl 2015;31:1466–8. https://doi.org/10.1093/bioinformatics/btu848.
[29] Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. J Youth Adolesc 1988;17:117–33.
[30] Martikainen S, Pesonen A-K, Lahti J, Heinonen K, Pyhälä R, Tammelin T, et al. Physical activity and hypothalamic–pituitary–adrenocortical axis function in adolescents. Psychoneuroendocrinology 2014;49:96–105. https://doi.org/10.1016/j.psyneuen.2014.06.023.
[31] Koskenvuo M, Hublin C, Partinen M, HeikkilÄ K, Kaprio J. Heritability of diurnal type: a nationwide study of 8753 adult twin pairs. J Sleep Res 2007;16:156–62.
[32] Montaruli A, Galasso L, Carandente F, Vitale JA, Roveda E, Caumo A. If the Morning-Evening Questionnaire (MEQ) is able to predict the actigraphy-based acrophase, how does its reduced, five-item version (rMEQ) perform? Chronobiol Int 2017;34:443–4. https://doi.org/10.1080/07420528.2017.1306708.
[33] Roveda E, Vitale J, Montaruli A, Galasso L, Carandente F, Caumo A. Predicting the actigraphy-based acrophase using the Morningness–Eveningness Questionnaire (MEQ) in college students of North Italy. Chronobiol Int 2017;34:551–62. https://doi.org/10.1080/07420528.2016.1276928.
[34] Vitale JA, Roveda E, Montaruli A, Galasso L, Weydahl A, Caumo A, et al. Chronotype influences activity circadian rhythm and sleep: Differences in sleep quality between weekdays and weekend. Chronobiol Int 2015;32:405–15. https://doi.org/10.3109/07420528.2014.986273.
[35] Olds TS, Maher CA, Matricciani L. Sleep Duration or Bedtime? Exploring the Relationship between Sleep Habits and Weight Status and Activity Patterns. Sleep 2011;34:1299–307. https://doi.org/10.5665/SLEEP.1266.
[36] Li W, Wu M, Yuan F, Zhang H. Sugary beverage consumption mediates the relationship between late chronotype, sleep duration, and weight increase among undergraduates: a cross-sectional study. Environ Health Prev Med 2018;23. https://doi.org/10.1186/s12199-018-0754-8.
[37] Vitale JA, Weydahl A. Chronotype, Physical Activity, and Sport Performance: A Systematic Review. Sports Med 2017;47:1859–68. https://doi.org/10.1007/s40279-017-0741-z.
[38] Lee JH, Kim SJ, Lee SY, Jang KH, Kim IS, Duffy JF. Reliability and validity of the Korean version of Morningness–Eveningness Questionnaire in adults aged 20–39 years. Chronobiol Int 2014;31:479–86. https://doi.org/10.3109/07420528.2013.867864.
[39] Wittmann M, Dinich J, Merrow M, Roenneberg T. Social Jetlag: Misalignment of Biological and Social Time. Chronobiol Int 2006;23:497–509. https://doi.org/10.1080/07420520500545979.
[40] Foti KE, Eaton DK, Lowry R, McKnight-Ely LR. Sufficient Sleep, Physical Activity, and Sedentary Behaviors. Am J Prev Med 2011;41:596–602. https://doi.org/10.1016/j.amepre.2011.08.009.
[41] Lang C, Kalak N, Brand S, Holsboer-Trachsler E, Pühse U, Gerber M. The relationship between physical activity and sleep from mid adolescence to early adulthood. A systematic review of methodological approaches and meta-analysis. Sleep Med Rev 2016;28:32–45. https://doi.org/10.1016/j.smrv.2015.07.004.
[42] Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. Sleep Health 2015;1:40–3. https://doi.org/10.1016/j.sleh.2014.12.010.

[43] Ohayon M, Wickwire EM, Hirshkowitz M, Albert SM, Avidan A, Daly FJ, et al. National Sleep Foundation's sleep quality recommendations: first report. Sleep Health 2017;3:6–19. https://doi.org/10.1016/j.sleh.2016.11.006.
[44] Davison KK, Werder JL, Trost SG, Baker BL, Birch LL. Why are early maturing girls less active? Links between pubertal development, psychological well-being, and physical activity among girls at ages 11 and 13. Soc Sci Med 2007;64:2391–404. https://doi.org/10.1016/j.socscimed.2007.02.033.
[45] Beal SJ, Grimm KJ, Dorn LD, Susman EJ. Morningness-Eveningness and Physical Activity in Adolescent Girls: Menarche as a Transition Point. Child Dev 2016;87:1106–14. https://doi.org/10.1111/cdev.12539.
[46] Duncan SC, Duncan TE, Strycker LA, Chaumeton NR. A cohort-sequential latent growth model of physical activity from ages 12 to 17 years. Ann Behav Med 2007;33:80–9. https://doi.org/10.1207/s15324796abm3301_9.
[47] Telama R, Yang X, Viikari J, Välimäki I, Wanne O, Raitakari O. Physical activity from childhood to adulthood. Am J Prev Med 2005;28:267–73. https://doi.org/10.1016/j.amepre.2004.12.003.
[48] Biddle SJ, Gorely T, Stensel DJ. Health-enhancing physical activity and sedentary behaviour in children and adolescents. J Sports Sci 2004;22:679–701. https://doi.org/10.1080/02640410410001712412.
[49] Merikanto I, Lahti T, Seitsalo S, Kronholm E, Laatikainen T, Peltonen M, et al. Behavioral trait of morningness-eveningness in association with articular and spinal diseases in a population. PloS One 2014;9:e114635.
[50] Merikanto I, Lahti T, Puolijoki H, Vanhala M, Peltonen M, Laatikainen T, et al. Associations of Chronotype and Sleep With Cardiovascular Diseases and Type 2 Diabetes. Chronobiol Int 2013;30:470–7. https://doi.org/10.3109/07420528.2012.741171.

Figure legends

Figure 1. Actigraph-based physical activity (PA, counts per minute, CPM) in girls and boys at T1 and T2 a) general PA per day and b) mean percentage of sedentary behavior, LPA and MVPA per day. Repeated Measures ANOVA p-values in Figure 1a indicate a significant difference in general PA between T1 and T2 among boys.



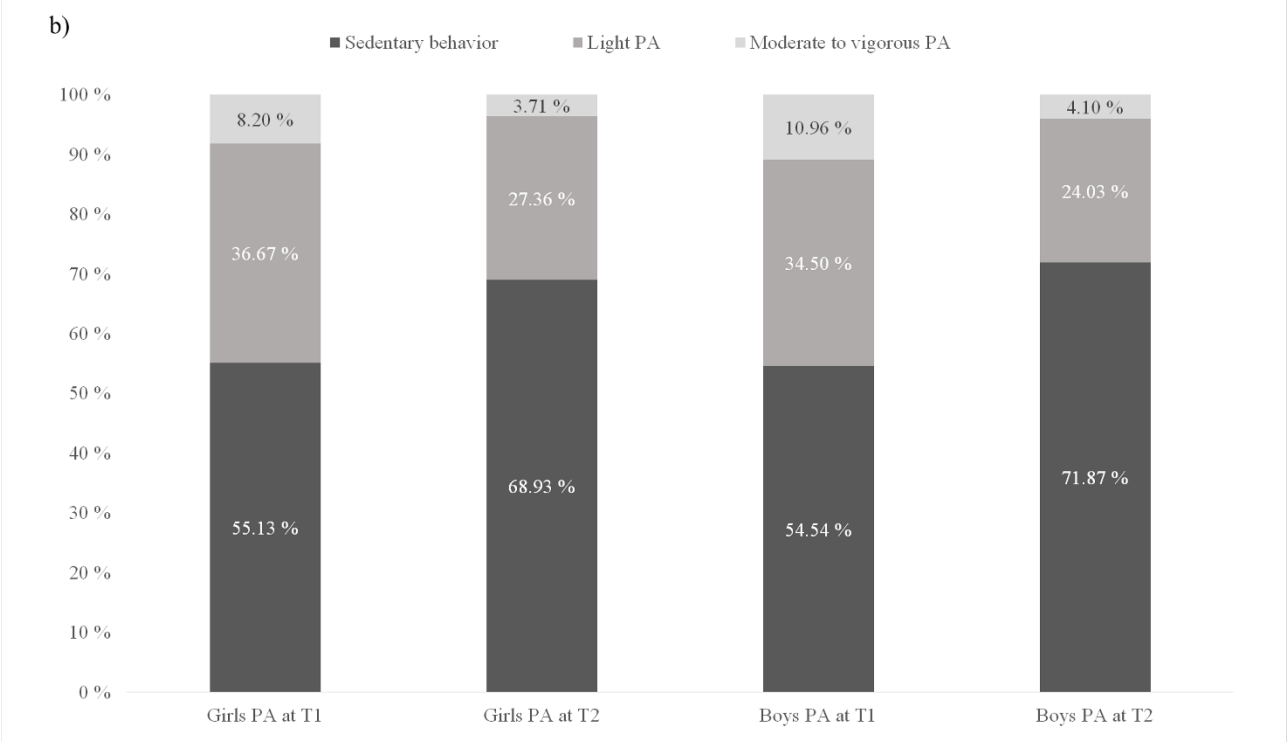


Figure 2. Change in general physical activity from T1 to T2 in three groups according to self-reported circadian preference (higher scores indicating higher morningness preference).

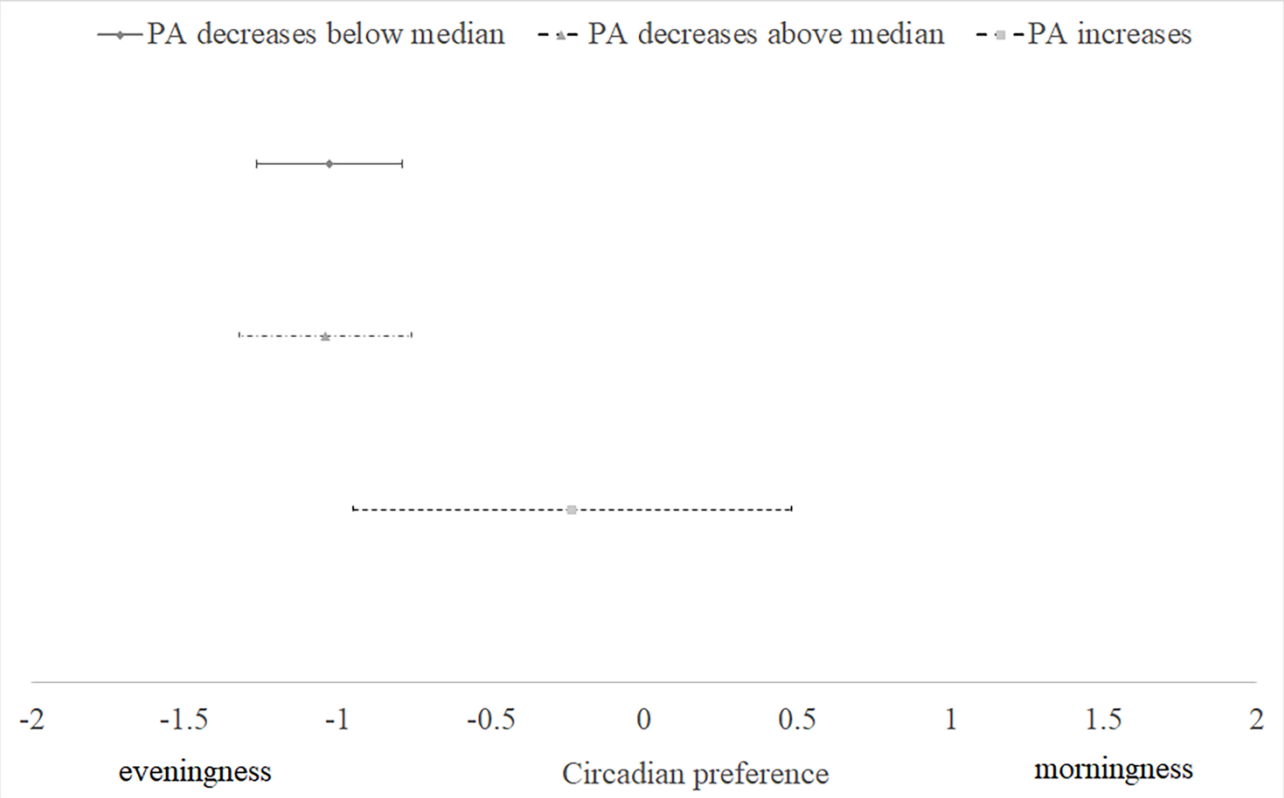


Table 1. Descriptive statistics of the sample by measurement points at T1 and T2.

	T1			**T2**		
	Girls (N = 187) Mean (SD)	**Boys (N = 166) Mean (SD)**	**P-value**	**Girls (N = 98) Mean (SD)**	**Boys (N = 73) Mean (SD)**	**P-value**
Age	12.32 (0.56)	12.35 (0.52)	0.63	16.89 (0.12)	16.89 (0.12)	0.98
PDS	2.04 (0.48)	1.55 (0.38)	6.3×10^{-22}	3.45 (0.26)	3.00 (0.38)	1.4×10^{-15}
BMI	19.54 (2.99)	19.42 (3.34)	0.71	22.26 (3.53)	21.67 (3.46)	0.27
Actigraphy usage days	7.85 (1.73)	7.89 (1.86)	0.83	6.65 (2.22)	7.26 (2.19)	0.07
General PA (mean cpm/day)	411.44(112.08)	451.95 (127.43)	0.002	281.50 (80.59)	272.40 (85.54)	0.48
Sedentary behavior (%/day)	55.13 (10.33)	54.54 (10.64)	0.60	68.93 (9.60)	71.87 (9.46)	0.04
LPA (%/day)	36.67 (6.64)	34.50 (6.86)	0.003	27.36 (7.68)	24.03 (7.91)	0.006
MVPA (%/day)	8.20 (5.21)	10.96 (6.06)	0.000006	3.71 (2.53)	4.10 (3.03)	0.37
Midpoint of sleep in hours:min	3:12 (0:37)	3:15 (0:42)	0.47	4:06 (1:05)	4:36 (1:18)	0.03
MEQ item 19 continuous	NA	NA	NA	-0.91 (1.18)	-0.95 (1.18)	0.85
Sleep duration in hours.min	8:00 (0:26)	7:54 (0:32)	0.06	7:08 (0:41)	6:54 (0:41)	0.03
Wake after sleep onset in hours:min	0:56 (0:16)	1:01 (0:20)	0.008	0:46 (0:17)	0:53 (0:24)	0.06

Note: PDS = Pubertal Developmental Scale, BMI = Body Mass Index, PA = physical activity, LPA = light PA, MVPA = moderate to vigorous physical activity, MEQ = Morningness/Eveningness Questionnaire.

Table 2. Correlation between circadian timing variables and physical activity (PA) at T1 and T2.

	Midpoint of sleep at T1		**PGS for morningness**		**Circadian preference at T2**		**midpoint of sleep at T2**	
	Pearson correlation *r*	**P-value**	**Pearson correlation *r***	**P-value**	**Pearson correlation *r***	**P-value**	**Pearson correlation *r***	**P-value**
T1								
PGS for morningness	-0.08	0.08						
General PA	-0.10	0.03	0.03	0.28				
Sedentary behavior	0.12	0.02	-0.05	0.19				
LPA	-0.10	0.04	0.07	0.13				
MVPA	-0.10	0.04	0.02	0.39				
T2								
PGS for morningness					0.16	0.03		
midpoint of sleep			-0.13	0.06	-0.33	0.00003		
General PA			0.06	0.26	0.19	0.01	0.02	0.40
Sedentary behavior			-0.04	0.33	-0.22	0.005	-0.03	0.37
LPA			0.04	0.34	0.21	0.006	0.03	0.37
MVPA			0.03	0.38	0.14	0.04	0.01	0.44

Note: PGS = polygenic score, LPA = light PA, MVPA = moderate to vigorous physical activity.

Table 3. Hierarchical Regression Model of actigraphy based physical activity (PA) at T1.

	General PA at T1			**Sedentary behavior at T1**			**LPA at T1**			**MVPA at T1**		
	R^2	R^2 *Change* (p-value[a])	β (p-value[b])	R^2	R^2 *Change* (p-value[a])	β (p-value[b])	R^2	R^2 *Change* (p-value[a])	β (p-value[b])	R^2	R^2 *Change* (p-value[a])	β (p-value[b])
Step 1 Variables entered: sex, age and pubertal status	0.10			0.07			0.06			0.13		
sex			0.11 (0.08)			0.01 (0.88)			-0.16 (0.02)			0.18 (0.006)
age at T1			-0.24 (0.00005)			0.24 (0.00006)			-0.19 (0.001)			-0.21 (0.0002)
pubertal status at T1			-0.11 (0.11)			0.08 (0.22)			-0.03 (0.71)			
Step 2 Variables entered: midpoint of sleep at T1	0.11	0.004 (0.24)		0.08	0.004 (0.28)		0.06	0.001 (0.58)		0.13	0.006 (0.16)	
sex			0.12 (0.07)			0.005 (0.94)			-0.16 (0.02)			0.18 (0.005)
age at T1			-0.22 (0.0002)			0.22 (0.0002)			-0.18 (0.003)			-0.20 (0.001)
pubertal status at T1			-0.11 (0.12)			0.08 (0.23)			-0.03 (0.71)			-0.12 (0.06)
midpoint of sleep at T1			-0.07 (0.24)			0.06 (0.28)			-0.03 (0.58)			-0.08 (0.16)
Step 3 Variables entered: PGS for morningness	0.11	0.001 (0.63)		0.08	0.002 (0.41)		0.07	0.004 (0.25)		0.13	0.00005 (0.90)	
sex			0.12 (0.07)			0.007 (0.91)			-0.16 (0.02)			0.18 (0.005)
age at T1			-0.22 (0.0002)			0.23 (0.0002)			-0.19 (0.002)			-0.20 (0.001)
pubertal status at T1			-0.11 (0.12)			0.08 (0.23)			-0.03 (0.72)			-0.12 (0.06)
midpoint of sleep at T1			-0.06 (0.26)			0.06 (0.32)			-0.03 (0.66)			-0.08 (0.17)
PGS for morningness			0.03 (0.63)			-0.05 (0.41)			0.07 (0.25)			0.007 (0.90)

Note: PA = physical activity, LPA = light PA, MVPA = moderate to vigorous physical activity, R^2 = Variance in dependent variable explained by the model, R^2 Change = Additional variance in dependent variable explained by the novel variable entered in the step, p-value[a] = Significance for additional variance in dependent variable explained by the novel variable entered in the step, β = Standardized coefficient, p-value[b] = Significance of the association between independent variable and dependent variable, PGS = polygenic score.

Table 4. Hierarchical Regression Model of actigraphy measured physical activity (PA) at T2.

	General PA at T2			**Sedentary behavior at T2**			**LPA at T2**			**MVPA at T2**		
	R^2	R^2 *Change* (p-value[a])	β (p-value[b])	R^2	R^2 *Change* (p-value[a])	β (p-value[b])	R^2	R^2 *Change* (p-value[a])	β (p-value[b])	R^2	R^2 *Change* (p-value[a])	β (p-value[b])
Step 1 Variables entered: sex, age and pubertal status	0.0002			0.02			0.04			0.02		
sex			-0.02 (0.88)			0.09 (0.41)			-0.14 (0.19)			0.09 (0.38)
age at T2			-0.001 (0.99)			-0.02 (0.81)			0.04 (0.66)			-0.04 (0.68)
pubertal status at T2			-0.002 (0.98)			-0.05 (0.66)			0.07 (0.52)			-0.03 (0.77)
Step 2 Variables entered: circadian preference	0.04	0.04 (0.02)		0.06	0.05 (0.01)		0.08	0.04 (0.02)		0.04	0.02 (0.07)	
sex			-0.05 (0.66)			0.12 (0.25)			-0.17 (0.10)			0.07 (0.52)
age at T2			0.01 (0.88)			-0.04 (0.68)			0.05 (0.54)			-0.02 (0.78)
pubertal status at T2			-0.07 (0.55)			0.02 (0.85)			0.004 (0.97)			-0.08 (0.46)
circadian preference			0.24 (0.01)			-0.22 (0.01)			0.21 (0.02)			0.16 (0.07)
Step 3 Variables entered: midpoint of sleep at T2	0.05	0.009 (0.27)		0.08	0.02 (0.14)		0.10	0.02 (0.11)		0.04	0.002 (0.56)	
sex			-0.07 (0.55)			0.14 (0.17)			-0.20 (0.06)			0.06 (0.58)
age at T2			0.007 (0.94)			-0.03 (0.75)			0.04 (0.61)			-0.03 (0.75)
pubertal status at T2			-0.07 (0.53)			0.03 (0.81)			-0.001 (0.99)			-0.08 (0.45)
circadian preference			0.24 (0.01)			-0.26 (0.004)			0.25 (0.005)			0.18 (0.06)
midpoint of sleep at T2			0.10 (0.27)			-0.13 (0.14)			0.14 (0.11)			0.05 (0.56)
Step 4 Variables entered: PGS for morningness	0.05	0.001 (0.67)		0.08	0.00006 (0.92)		0.10	0.00001 (0.97)		0.04	0.0003 (0.84)	
sex			-0.07 (0.54)			0.15 (0.18)			-0.20 (0.06)			0.06 (0.59)
age at T2			0.007 (0.93)			-0.03 (0.75)			0.04 (0.61)			-0.03 (0.75)
pubertal status at T2			-0.07 (0.51)			0.03 (0.81)			-0.001 (0.99)			-0.09 (0.44)
circadian preference			0.23 (0.01)			-0.26 (0.005)			0.25 (0.006)			0.18 (0.06)
midpoint of sleep at T2			0.10 (0.25)			-0.13 (0.14)			0.14 (0.12)			0.06 (0.55)
PGS for morningness			0.04 (0.67)			-0.008 (0.92)			0.004 (0.97)			0.02 (0.84)

Note: PA = physical activity, LPA = light PA, MVPA = moderate to vigorous physical activity, R^2 = Variance in dependent variable explained by the model, R^2 Change = Additional variance in dependent variable explained by the novel variable entered in the step, p-value[a] = Significance for additional variance in dependent variable explained by the novel variable entered in the step, β = Standardized coefficient, p-value[b] = Significance of the association between independent variable and dependent variable, PGS = polygenic score.